CHAPTER

8

Neonatal Cortical Rhythms

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OUTLINE

Nomenclature

AMPA α-Amino-3-hydroxyl-5-methyl-4-isoxazole-propionate
DC Direct coupled
EEG Electroencephalography
ENO Early network oscillations
GABA γ-Aminobutyric acid
GDP Giant depolarizing potential
LGN Lateral geniculate nucleus
MEG Magnetoencephalography
NMDA *N*-Methyl-D-aspartic acid
ODCs Ocular dominance columns
P Postnatal day
R Receptor
S1 Primary somatosensory cortex
SAT Slow activity transient
STDP Spike-time-dependent plasticity
V1 Primary visual cortex
μV Microvolt

8.1 INTRODUCTION

The fetal period in humans is characterized by a number of fundamental events in the construction of the nervous system, such that at birth, many of the primary circuits already have been formed and display remarkable functional performance, although development evidently continues after birth until full maturity is reached at around age 30. Considerable evidence indicates that electrical activity expressed in the human fetal brain – and in lower mammals at corresponding developmental stages – controls a number of developmental processes, including neuronal differentiation, migration, synaptogenesis, and synaptic plasticity (for review, see Ben-Ari et al., 1997; Blankenship and Feller, 2010; Feldman et al., 1999; Feller and Scanziani, 2005; Fox, 2002; Henley and Poo, 2004; Katz and Crowley, 2002; Katz and Shatz, 1996; Rakic and Komuro, 1995; and Zhou and Poo, 2004a). Probably the most thoroughly elaborated evidence has been generated by studying sensory cortices, in which development of sensory maps is critically influenced by activity from the sensory periphery. However, the physiology of the fetal central nervous system, and notably the electrical patterns of organized neuronal activity that underlie map formation, has remained obscure for a long time. This is mainly a result of technical limitations in recording electrical activity from the fetal brain in utero. An important and almost paradoxical aspect of the problem is that the fetus develops

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